# Visualizing the EKG

## Introduction

You do NOT have to become an expert interpreter of the electrocardiogram (we intentionally flip between EKG and ECG because both are used so often in practice). You do NOT need to understand lead vectors. You do NOT have to visualize the heart as seen from 12 different cameras. If you don't know what that is referring to, then good, we hope you never do. Unless you are going to be an electrophysiology-subspecialized cardiologist (internal medicine residency for 3 years, cardiology fellowship for 3 years, then a 1–2-year extra fellowship in EP), there are very few things you need to be able to do with an EKG. The EKG provides more information than this lesson will teach you. The thing is that an ECG provides the **diagnosis** of the heart's rhythm (arrhythmias) and the **diagnosis** of total occlusion of a coronary vessel (ST-segment elevation myocardial infarction). Beyond that, the EKG only provides **clues** of an underlying diagnosis or characteristic of a patient's heart. The underlying diagnosis or characteristic is better assessed with a different tool (echocardiogram, angiogram), so we don't want you to get caught up in all the features of an EKG.

In this lesson, we show you what the electrocardiogram is and how the cardiac conduction system maps onto an EKG tracing. We go over the basics, naming the waves and the lines, telling you what they mean. The goal is to see atrial electrical systole and diastole, ventricular electrical systole and diastole, and how the cardiac conduction system—SA node to AV node, AV node to bundles to apex, apex back up to Purkinje fibers—can be seen with each heartbeat. We absolutely do NOT want you to try to map the cardiac action potential with the EKG. The **EKG measures the mass of electrical change**. The cardiac action potential is also electrical—charges, depolarizations, Sodium Channels, Calcium channels, Potassium Channels—but independent of the electrical tracing of the EKG. The size of the tissue depolarizing—not how powerful the action potential is—determines the amplitude of the trace on the EKG.

A **rhythm strip** is the printout of a single lead. It is a continuous recording of the heart events and usually has a few heartbeats on it. To obtain a rhythm strip, you look at one lead of a 3-lead ECG. Leads are not electrodes. Leads are recording vectors based on the placement of three recording leads and a grounding wire. Just kidding. No vectors. A rhythm strip is used to identify the rhythm. The rhythm strip can be on a 12-lead EKG readout or on its own.

A **12-lead ECG** is useful for identifying myocardial infarction. A 12-lead has 12 short rhythm strips on it, each with only a few beats. Below the 12 leads, there will be one long rhythm strip. The rhythm strip is used to identify the rhythm, and the 12 leads are used to identify ST-segment changes.

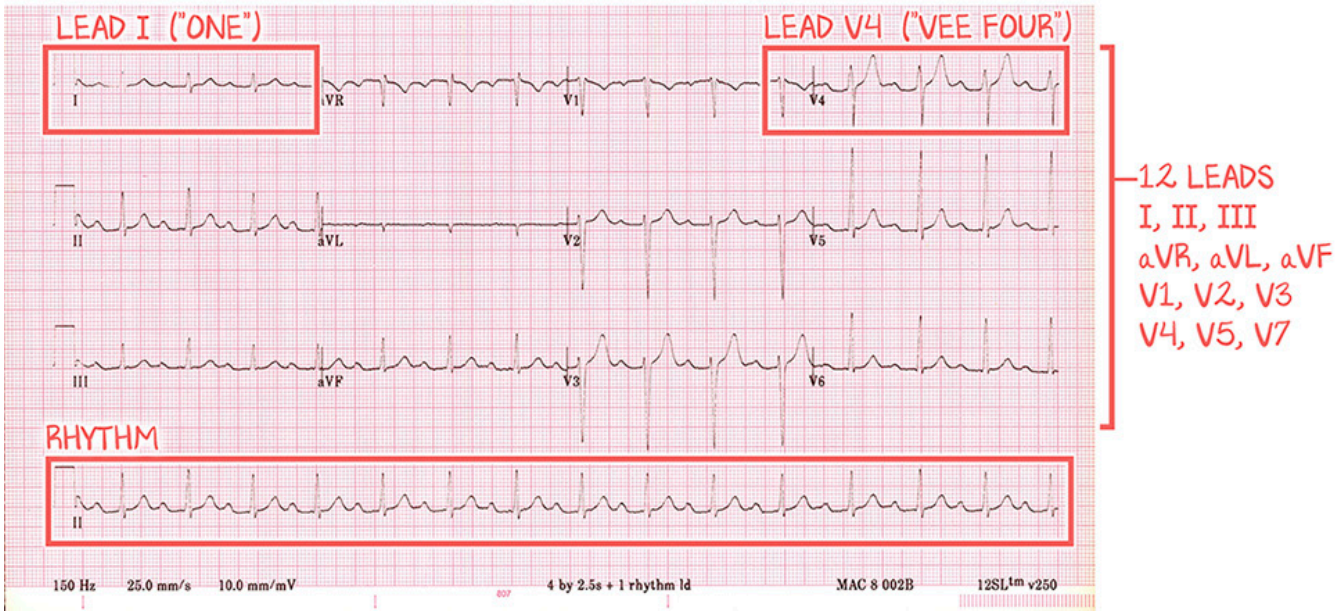


**Figure 3.1: The EKG**

## From Depolarization to EKG

An EKG visualizes changes in electrical activity. It reads the depolarization and repolarization of cardiac tissue. The contraction of the ventricle will follow the depolarization. The EKG cannot see the contraction. It cannot see relaxation. **The EKG can only show depolarization and repolarization.** That means we cannot tell how hard the contraction is or how much volume is expelled.

There is a P-QRS-T nomenclature applied to EKGs. This is the way medical science named bumps and spikes on an EKG. If there was a special reason for those particular letters, there isn't one now. The letters don't stand for anything. They are not an advanced organizer. But they name the waves and deflections in the order they occur. The P wave comes first, the QRS complex after, and the T wave last.

The **x-axis** of an EKG shows **time**. Depolarization through the conduction system takes time. Standard readouts of ECGs divide the strip into boxes. One big box is five little boxes. Each little box is 40 ms. One big box is 200 ms. We know normal conduction starts in the atrium, travels down through the AV node, then through the interventricular septum to the apex of the heart, then back up the lateral walls of each ventricle. We are able to use the EKG to follow that signal over time.

The **y-axis** measures height in millimeters. Height is how the EKG measures the magnitude of polarity change—either depolarization or repolarization. Be careful, the ECG height is NOT a product of how big a change in potential any one cell is experiencing. Those are electrical changes, and we will address the "tracings" of individual myocyte action potentials in the next lesson. Those tracings are not on an ECG. The height of a tracing on an EKG is a measurement of the **mass of tissue that is changing polarity**. For example, there are lots of myocytes in the ventricles, and therefore when the ventricles do something, the deflections are large, whereas there are not a lot of myocytes in the atrium, so when the atrium does something, the deflection is smaller. The P wave is tiny, and the QRS complex is huge. The more tissue there is, the more depolarizing cells there are, the more of a signal the EKG machine gets, the **higher the waveform** rises.

Then there are deflections (waves and complexes), segments, and intervals. We want you to see it simply first, and then we'll trace the conduction system through the heart, then map it back to the ECG.

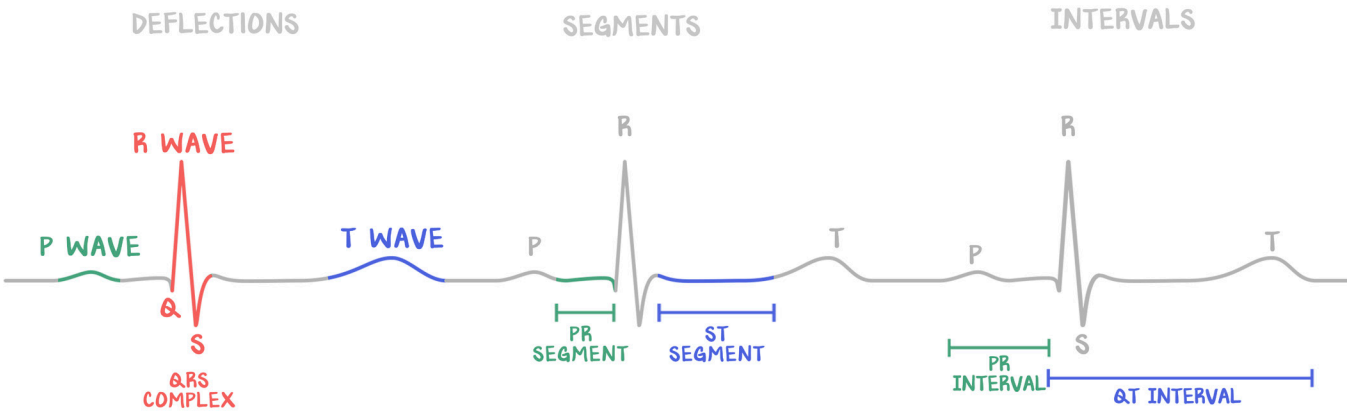


**Figure 3.2: The EKG**

We start at the SA node. It depolarizes, initiating the depolarization of the atria. But the entirety of the atria doesn't depolarize at the exact same time because the depolarization must travel through the syncytium. The fewest myocytes depolarize at the beginning and the end of the depolarization. There is a gradual increase in the mass of depolarization until it peaks somewhere in the middle of the atrium. Then there is a gradual decrease in the mass of depolarization as it finishes. The **P wave** is **atrial depolarization**.

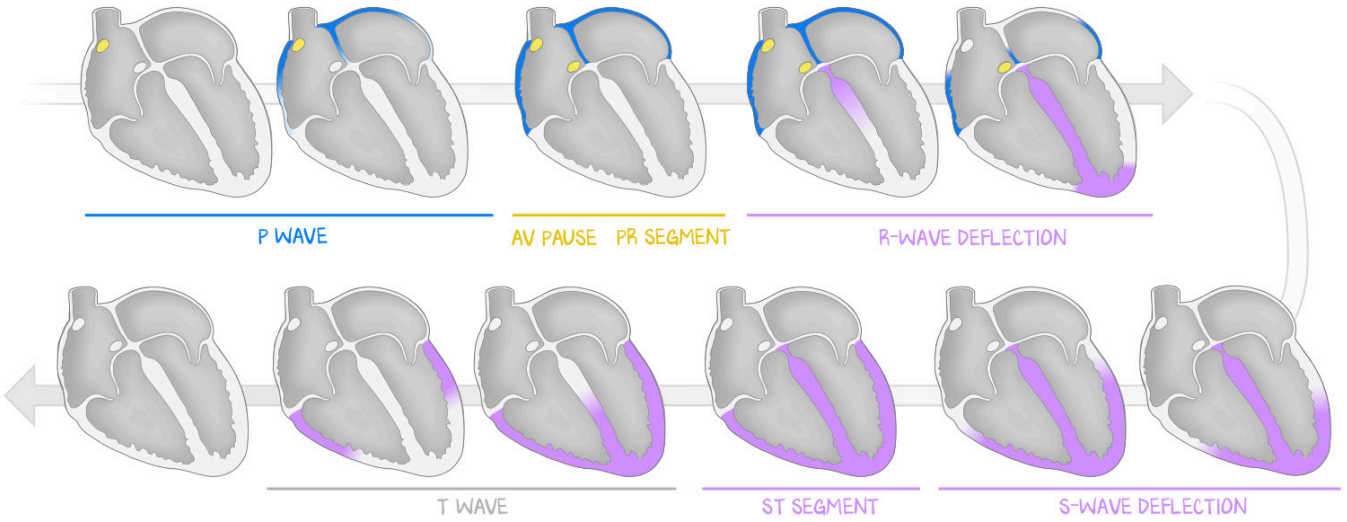


**Figure 3.3: Visualizing the Surrounding Paragraphs**

The signal reaches the AV node. There, conduction velocity is slow. The atria have depolarized, and the ventricles have yet to depolarize. The AV node is small. Because it is so small, the depolarization of AV node myocytes cannot be detected by the EKG. Without any depolarization or repolarization, the EKG returns to the **isoelectric line** (its baseline). The **PR segment** is the time the signal is conducting (slowly) through the AV node. It is also period wherein the atria contract to inject the atrial kick into each atrium's ventricle – the excess preload better optimizing the sarcomere length of the ventricle, excess perfusion pressure.

The signal exits the AV node into the bundle of His, and is then conducted by the bundle branches. The direction of depolarization is down toward the apex within the interventricular septum. A large mass of myocardium is depolarizing toward the apex. The **R wave** (the first upward deflection of the QRS complex) is the signal traveling down toward the apex. The signal is then propagated up the side of each ventricle's lateral aspect on Purkinje fibers. The **S wave** (the negative deflection of the QRS complex that follows the R wave) is the signal traveling away from the apex. Somewhere within the QRS complex, the atria repolarize. Because the ventricular syncytium has so many cells depolarizing, the atrial repolarization is not seen.

Just as with atrial depolarization and contraction, after ventricular depolarization (after the QRS), cells are neither depolarizing nor repolarizing. The EKG detects no change in status, so it returns to the **isoelectric line**. As the ventricle repolarizes, the EKG detects a change. The wave of depolarization started at the interventricular septum, down to the apex, and back up the sides. The wave of repolarization does the same. Just as the depolarization was an upward deflection traveling down toward the apex, so too is repolarization. This confuses learners because depolarization and repolarization are opposite electrical phenomena, yet the **T wave** is supposed to be a positive deflection above the isoelectric line. We've really simplified things to avoid vectors, leads, and the like. The **direction of travel** the electrical impulse is following determines whether it is a positive or negative deflection. You can assume, at this stage in your training, that *down toward the ventricle* is an upward deflection and *up the sides from the apex* is a downward deflection.

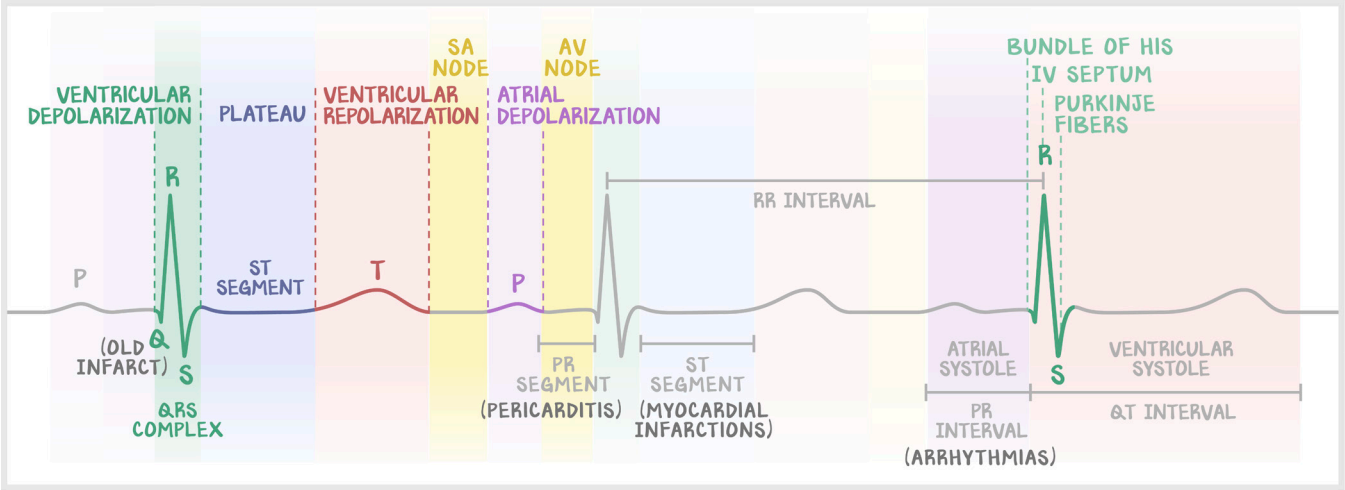


**Figure 3.4: Putting It All Together**
This combines Figure 3.2 and Figure 3.3. You don't have to memorize this figure. It is overly complicated, but presents several concepts mapped together. Here you see the intervals and segments mapped onto what you learned while studying the cardiac cycle in the Plumbing island. Here, we also give you a preview of the clinical relevance of the deflections, segments, and intervals. A Q wave is pathologic, a sign there had been an infarct, and is often not seen. The PR segment is useful in diagnosing pericarditis. The ST segment is used to identify myocardial infarctions (more on this next). The PR interval is used to diagnose heart blocks (next lesson). The QT interval (also called the QTc) is used as a marker of proarrhythmic side effects of medications. Finally, the RR interval, the distance between QRS complexes, is used to determine the heart rate (next lesson).

## Interpreting the 12-Lead

12-Lead ECG requires many electrodes to get all the right angles. Each lead is a perspective of the heart, and certain areas of the heart are best evaluated by certain leads. Twelve seems like a lot. But we can compartmentalize it for you.

12-Leads also come with a rhythm strip. You look at the rhythm strip, the 3-lead, for rhythm interpretation. If the 12 leads scare you, fold over the printout so that you see only the one lead. Do all the things we teach you in the next lesson—figure out what the rhythm is. Then unfold the paper and use the 12-lead for what is designed to do—identify ST-segment elevation myocardial infarction. Don't attempt to use the 12 leads for rhythm interpretation.

We are about to teach you how to look at the 12-lead to look for ST-segment changes, axis deviation, and left ventricular hypertrophy. CAN you look at the 12-lead for more than that? Yes. Will you? Someone is more than likely going to make you.

## Identifying Coronary Arteries with the 12-Lead

In order to diagnose an ST-elevation myocardial infarction (STEMI), 100% occlusion of a coronary vessel, there must be ST-segment elevations in two anatomically contiguous leads with reciprocal changes in other leads. That's a mouthful. First, note that it is **anatomically contiguous** and not contiguous on the 12-lead readout. If there are ST-segment changes in one anatomic region, there will be ST-segment depressions somewhere else. Unless you go into emergency medicine or cardiology, you will not have to do more than this. On an exam, the test isn't going to sneak by a STEMI. The goal is to assess your knowledge of coronary anatomy and how to read an OBVIOUS EKG. So, let's keep it at a birds-eye view. We are NOT going into copious technical detail.

The **inferior leads** are anatomically contiguous. They give you information about the **inferior heart**. The inferior heart is the lateral wall of the **right ventricle**. They are limb leads II, III, and aVF (say "*limb leads two, three, and avf")*. The territory monitored by the inferior leads is fed by the **right coronary artery** and its marginal branch.

The **lateral leads** are anatomically contiguous. They give you information about the **lateral heart**. The lateral heart refers to the left lateral heart, the lateral wall of the left ventricle. They are leads I, $V_5$, and $V_6$ (say "*Leads one, vee five, and vee six*"). The territory monitored by the lateral leads is fed by the **left circumflex artery** and its marginal branch.

The **anterior leads** are anatomically contiguous. They give you information about is the **anterior heart**. The most anterior structure within the mediastinum is the right ventricle, but the anterior heart refers to the anterior descending artery within the interventricular groove, not the most anterior chamber. Thus the anterior leads provide information of the **left ventricle's anterior**. They are leads $V_1$, $V_2$, $V_3$, and $V_4$ (say "*leads vee one through vee four")*. The territory monitored by the anterior leads is fed by the **left anterior descending artery**.

The **septal leads** are anatomically contiguous. They give you information about the **interventricular septum**. The septum is in the middle of the human heart, between the two ventricles. The septal leads are leads $V_1$ and $V_2$. Their territory is fed by a branch of the left anterior descending, the **septal branch**.

Commit inferior leads, lateral leads, anterior leads, and septal leads to memory. There are others, and only one deserves any mention—posterior leads.

There are only **posterior leads** if you place the electrodes those leads require. Placing EKG electrodes on a person's back isn't common, but it is required to view the posterior leads. However, because there is semi-symmetry to the adult heart, and the anterior leads correspond to the left anterior descending artery, the posterior leads evaluate the **posterior descending artery** and the perfusion of the **posterior left ventricle**. Therefore, due to the way $V_1$–$V_4$ are oriented, they also detect the posterior leads, but backward. So ST-segment depression in leads $V_1$–$V_4$ may be ST-segment elevation in leads 13, 14, and 15, the posterior leads.

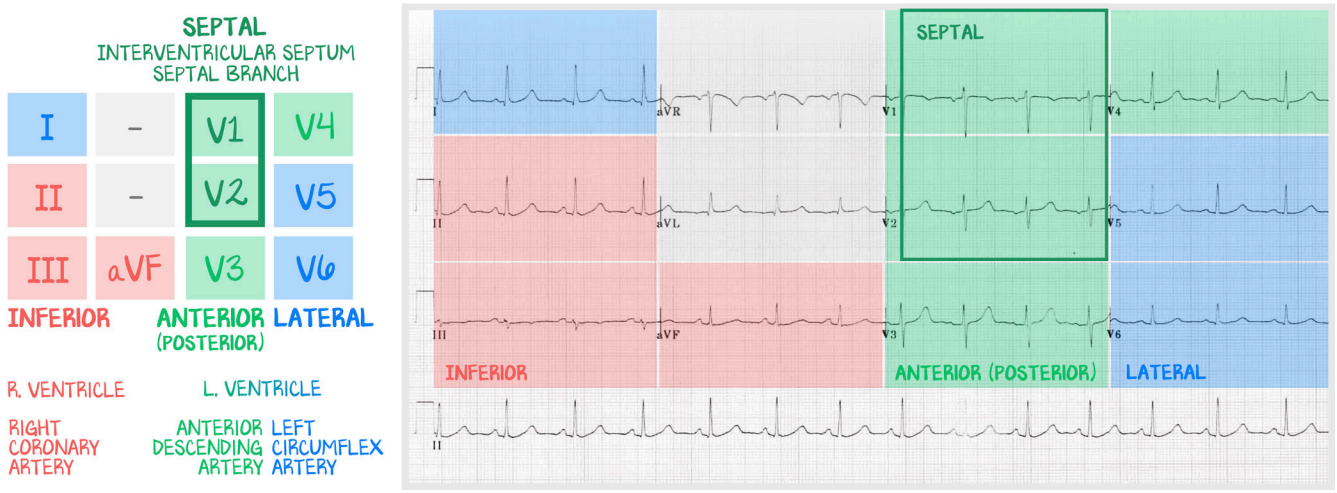


**Figure 3.5: 12-Lead to Coronary Artery**
If there are ST-segment elevations in two or more anatomically contiguous leads, the diagnosis of ST-segment elevation myocardial infarction (complete occlusion of a coronary vessel) is diagnosed. This is a representation of the anatomically continuous leads, identified by color.

## Using the T Wave to Deduce the Damage

The T wave can mean a lot of things. When the T-wave changes are in anatomically contiguous leads, they mean what we're about to tell you.

**T-wave inversion** means myocardial ischemia. Whether it be demand (strain) or supply (partial occlusion) ischemia, the myocardium is injured. It means that, at the very least, there is a subendocardial (not transmural) infarction. T-wave inversions are the least specific finding. But finding them means something acute is going on with the heart.

**ST-segment elevation** means **infarction**. Infarction means the tissue isn't dead yet but will be soon. ST-segment elevations signify a transmural myocardial infarction: from the epicardium to the endocardium, all the myocytes are dying. Blood supply has been cut off to a segment of the myocardium. A coronary artery is blocked. It is still early, though; none of the myocytes are dead yet. They will die if you don't do something. But ischemia only results in irreversible necrosis after 4 hours. For the first 4 hours, reperfusion can save the myocytes at risk.

**ST-segment elevations with a Q wave** means heart myocytes are actively dying and that some are dead. The ST-segment elevation means that there is tissue to save. The Q wave means that there is tissue beyond saving. A Q wave is any negative deflection at the beginning of a QRS complex. It is called QRS because each line is part of ventricular contraction, but the Q wave isn't supposed to be in every QRS complex—Q waves are pathologic. Without any ST-segment elevation, there is no good heart to save. The infarct happened. The Q wave will forever be there.

**Q waves only** mean an old infarct. They will be in the vascular distribution of the original infarct. They exist because there are dead myocytes, granulation tissue, or scar. None of those things is a myocyte. None of those things will depolarize. The Q wave represents **abnormal depolarization**.

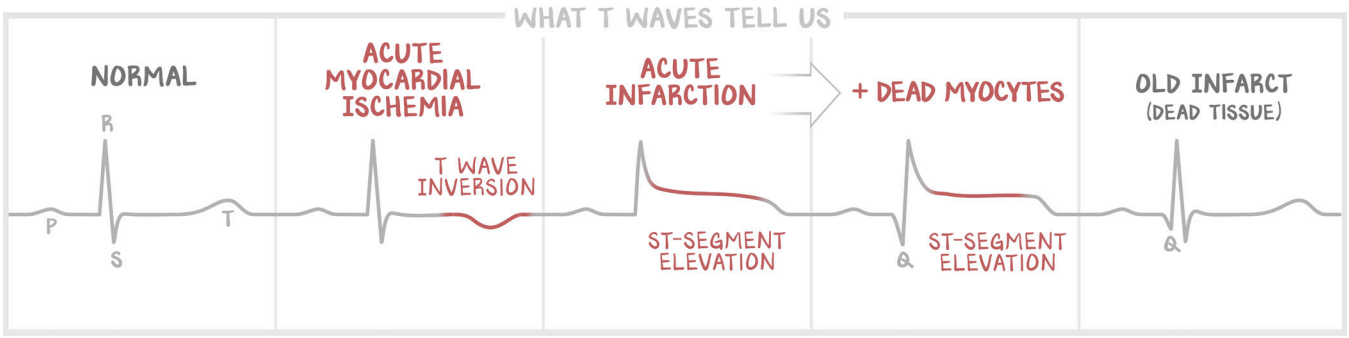


**Figure 3.6: What the T Waves Tell Us**

## Using the 12-Lead to Interpret Axis Deviation

Axis deviation has a place in interpreting the 12-lead for clues of other diseases. Unless your choice of specialty demands it of you, you won't need more than this. We use the **rocket-ship method**. Leads I and aVF are the two leads you will use. Now actually do this in front of you, right now, as you go. Give yourself a big thumbs up, both hands. Lift your left hand a little higher than your right. Now put your hands down, you look ridiculous. Look at the 12-leads in Figure 3.5. Find Leads I and aVF.

Leads I and aVF . . . hey . . . lead I is a little higher up than aVF on the page. You didn't look ridiculous, put your thumbs back up, double thumbs-up, left hand a little higher than the right. **Two thumbs up = normal axis.**

Tilt your thumbs out a little. Your thumbs are rocket ships, and the tips of your thumbs are the front of the ship, the direction of travel. The rockets will only fire if they are in the up position. Click, both fire straight up you go, normal axis.

Back to the starting point, both thumbs up, the right lower than the left. Turn your right hand, thumbs down. Ignite the rockets. Only the left fires, and up and to the left you go. That's **left axis deviation**.

Return your thumbs to both thumbs up. Now turn your left hand, thumbs down. Ignite the rockets. Only the right fires, and up and to the right you go. That's **right axis deviation**.

Put both thumbs down. Boooooooo. Sad double thumbs down. That's the only other option there is. **Extreme left axis deviation**.

To translate: the thumb direction is the QRS complex. If the deflection above the isoelectric line is larger than the deflection below the isoelectric line, then it is positive, thumbs up. If the deflection below the isoelectric line is larger than the deflection above the isoelectric line, then it is negative, thumbs down.

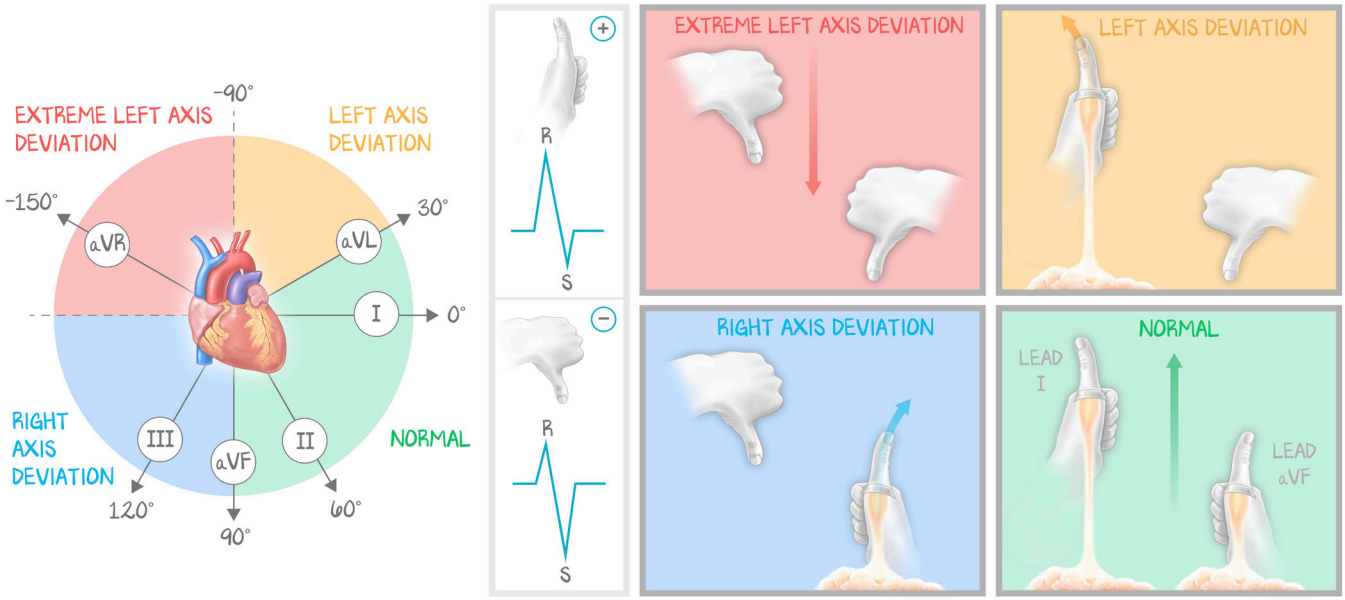


**Figure 3.7: Axis Deviation**
How the axis is determined for real does not look fun. It isn't. Don't do it. No vectors, only rocket ships! Summary of the rocket-ship method. Up means that the R wave is taller than the S wave is deep. Down means the S wave is deeper more than the R wave is tall. The thumb method was illustrated to match the classic vector approach, where the heart points down and to the right (as if you were looking at a patient head-on).

## Assessing Ventricular Hypertrophy

There are several ways to do this. The most effective on test day is, *"are any of the QRS complexes in V1–V6 massively enlarged whereas the others aren't? Yes = hypertrophy, No = no."*

The second most effective method on test day is the **inappropriate touching technique**. If inappropriate touching of neighboring QRS complexes occurs in the V leads, if they start running into each other in the V leads but not anywhere else, you've got hypertrophy. If they are touching everywhere, it is likely because the voltage is turned up too high, and you cannot use the inappropriate touching technique.

The slightly more technical way of doing it is to use **voltage criteria**. There are several. The one we recommend is [$V_1$ or $V_2$] + [$V_5$ or $V_6$]. Find the deepest, most negative QRS in $V_1$ or $V_2$. Find the tallest, most positive QRS in $V_5$ or $V_6$. Count the number of small boxes. Add them together. If that number is greater than 35, you have hypertrophy.

A much, MUCH better way to assess for ventricular hypertrophy is point-of-care ultrasound. Put the probe in parasternal, short axis, snap a picture, and measure the thickness of the ventricular wall. The best way to determine whether there is ventricular hypertrophy is to have a trained radiology technician perform a formal echocardiogram and have a cardiologist interpret said echocardiogram. Voltage criteria for hypertrophy are only a clue for hypertrophy and are not diagnostic. This is why *"are they big?"* and *"are they touching?"* are reasonable techniques.

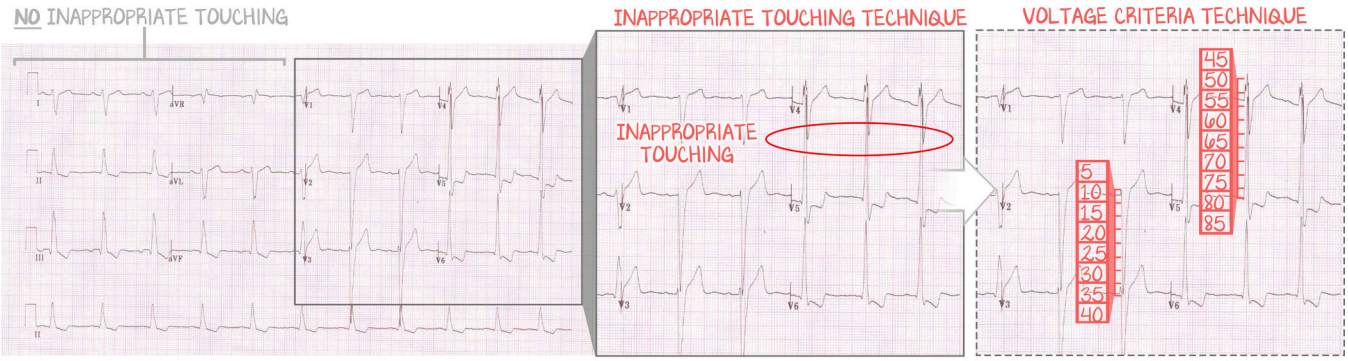


**Figure 3.8: Left Ventricular Hypertrophy**

## Citations

Figure 3.1: Courtesy of Mike Cadogan, M.D. LITFL.com.

Figure 3.8: Courtesy of Mike Cadogan, M.D. LITFL.com.